Exhibit 12

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Specific Causation Expert Report: Frank W. Mousser

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VIII. Kidney Cancer Risk Associated with TCE

The International Agency for Research on Cancer (IARC) classifies Trichloroethylene (TCE) as a human carcinogen, specifically citing "sufficient evidence in humans for the carcinogenicity of trichloroethylene. Trichloroethylene causes cancer of the kidney."[2] In addition, available evidence has provided a cohesive database supporting TCE as a known kidney carcinogen. This has been demonstrated in both human and animal studies, with mechanistic data suggesting that the carcinogenic effect of TCE results from its metabolism into genotoxic and cytotoxic intermediates that target the kidney and cause DNA strand breaks and mutations in tumor suppressor genes. The relationship between TCE exposure and kidney cancer risk has been documented in direct occupational exposure as well as residential chronic exposure at low to moderate doses. A study examining kidney cancer risk associated with historic groundwater contamination revealed the 50^{th}-75^{th} percentile of estimated exposure over a 15 year period was associated with an increased risk of kidney cancer with adjusted odds ratio (OR) of 1.78 95% confidence interval (CI) compared to $<50^{th}$ percentile.[3] In this study, the maximum measured groundwater TCE levels varied widely, with estimated TCE exposure levels generally ranging from 0-27.6 ug/L.[3]

Another study providing epidemiologic evidence supporting the association between TCE and renal cell carcinoma risk examined occupational TCE exposure in several European countries.[4] TCE exposure was categorized into one of three levels ranging from 0-<27ug/m^3, 27-270 ug/m^3 and >270 ug/m^3, with almost all TCE exposure occurring at least 20 years before disease onset.[4] For TCE exposure, ORs were significantly elevated for all exposure indices (OR = 1.63-2.34).[4] In addition, this study examined the association between TCE exposure and renal cell carcinoma risk after stratification by GSTT1 genotype, which revealed significant associations among subjects exposed to TCE with an active genotype (OR 1.88; 95% CI) but not among GSTT1 nulls (OR 0.93, 95% CI).[4] The findings of this study support the genotoxic mechanism believed to be causative in the development of renal cell carcinoma in these cases. A follow up analysis examined the association between TCE exposure and subtypes of clear cell renal cell carcinoma, with clear cell B subtypes demonstrating a statistically significant elevated measure of association (OR 3.09).[5]

Additional studies include Karami et al (2012) which also demonstrated that TCE can cause kidney cancer, as the authors performed a meta-analysis of 9 cohort studies which resulted in an overall elevated relative risk of 1.26 (95% CI 1.02-1.56) for TCE exposure and renal cancer.[6] Another meta-analysis included 23 studies: 16 cohort and 7 case-control.[7] This study demonstrated significantly elevated measures of association across all studies (RR 1.42), in only case-control (RR 1.33), and in only studies with well documented exposure assessment (RR 1.34).[7]

In addition to these references, there is literature directly relating to the toxins in the water at Camp Lejeune that supports the causal association between TCE and kidney cancer. Bove et. al. 2014a specifically studied the toxins in the water at Camp Lejeune and found associations between the Camp Lejeune water with all the chemicals at issue (TVOCs) and also individual chemicals.[8] Bove et. al. 2014a found a monotonic exposure response for TVOCs at Camp Lejeune relating to kidney cancer with RR of 1.42 (low exposures), 1.44 (medium exposures) and 1.54 (high exposures).[8] The supplemental tables in this study specifically detail HR for cumulative exposures to TCE for the individuals exposed at Camp Lejeune.[8] The HR for cumulative exposures to TCE were 1.54 (low exposures), 1.21 (medium exposures) and 1.52 (high exposures).[8]

There were additional causal relationships found between the toxic water at Camp Lejeune/TCE in the water at Camp Lejeune and kidney cancer. For example, Bove 2024 (both cancer incidence and cancer mortality) support a causal association for individuals exposed to the water at Camp Lejeune and kidney cancer. [9,10]

Finally, just recently, the EPA gave public notice of a final rule change completely banning TCE in the United States.[11] In the public notice of EPA's ban of TCE, the EPA and its spokespeople specifically listed the connection between TCE and kidney cancer as a reason for the need for the ban.[11] In its notice and rule, it cited Camp Lejeune's water contamination as an example of how TCE can cause cancers, including kidney cancer, at low levels.[11]

I have read the general causation report of experts Dr. Benjamin Hatten and Dr. Steven Bird. These expert reports detail an extensive review of the epidemiology, toxicology and mechanism of action of TCE and kidney cancer. These reports are consistent with my review of the literature and support my opinions in this case.

Of note, Dr. Hatten's report states,

Given that non- parenchymal upper urinary tract (renal pelvis and ureter) shares blood supply from the renal artery with the body of the kidney and that urine flows from the kidney through the renal pelvis and ureter it is at least as likely as not that urothelial cancers share the described carcinogenic mechanism with kidney cancers.

IX. Kidney Cancer Risk Associated with PCE, VC and Benzene

The IARC has classified both vinyl chloride (VC) and benzene as known human carcinogens and PCE as "probably carcinogenic to humans."[2,12] Available epidemiologic data is consistent with toxicological evidence of PCE's carcinogenicity.

a. PCE

Mechanistically, PCE is thought to induce kidney cancer via genotoxicity, oxidative stress leading to DNA strand breaks and mutations, and direct cellular cytotoxicity. Epidemiologic studies involving PCE exposure demonstrate an association with kidney cancer. Aschengrau *et al.* reviewed the cancer risk experienced by a cohort of individuals exposed to PCE via contaminated water supplies on Cape Cod, Massachusetts.[13] Following this discovery, the Massachusetts Department of Health observed "elevations in cancer mortality" in affected areas.[13] This population was then matched to population-based controls to define the risk of cancers for the Cape Cod cohort.[13] The authors found that any PCE exposure (OR 1.23) and low PCE exposure (OR 1.36) demonstrated elevated measures of association with kidney cancer in an analysis not accounting for latency.[13]

The 2018 ATSDR Morbidity Study of Marines and civilians at Camp Lejeune found there was a monotonic exposure-response relationship between kidney cancer risk and TCE/ PCE exposure for Marines.[14] ORs were ≥1.5 for both TCE and PCE in Marines and for TCE/PCE in civilian employees.[14] In addition, an occupational case-control study published after the ATSDR Assessment reported an OR of 3.0 (95% CI: 0.99, 9.0) for kidney cancer among those with high PCE exposure intensity and high cumulative exposure after excluding those with ≥50% probability of TCE exposure.[15]

Many studies examining PCE exposure in occupations involve the dry-cleaning industry. For example, an elevated measure of association (SMR 1.41) for kidney cancer mortality was reported in a cohort study of dry cleaner union members who worked in PCE exposed shops for at least a year prior to 1960 with up to a 20-year latency period.[16]

Further, the EPA just enacted a rule banning PCE products and in that rule used as a basis that PCE is causally associated with kidney cancer and that PCE can cause kidney cancer at low levels.

b. Vinyl Chloride

Mechanistically, vinyl chloride is thought to induce kidney cancer via oxidative stress leading to DNA strand breaks and mutations and the formation of DNA adducts. A DNA adduct is a segment of DNA that is chemically bonded to a cancer-causing chemical, inducing carcinogenesis.

There are epidemiologic studies involving vinyl chloride exposure that demonstrate an association with kidney cancer. Hu et el al (2002) demonstrated an increased risk of renal cell carcinoma in males with occupational exposure to vinyl chloride, in a dose-response manner, with the excess risk being significantly associated to duration of exposure.[17] Compared with no exposure to vinyl chloride, the adjusted OR was 2.0 (95% CI = 1.2–3.3).[17] In addition, Bove at al (2014a) found an elevated measure of association (HR 1.55) for kidney cancer deaths of military personnel stationed at Camp Lejeune compared to Camp Pendleton with at least low exposure to vinyl chloride.[8] Bove et al (2014a) found significantly increased HR at low, medium and high levels of exposure to VC; 1.66 (low exposure), 1.61 (medium exposure) and 1.51 (high exposure).[8]

c. Benzene

Mechanistically, benzene is thought to induce kidney cancer via its metabolites inducing oxidative stress leading to DNA strand breaks and mutations and the formation of DNA adducts.

There are epidemiologic studies involving benzene exposure that demonstrate an association with kidney cancer. The Hu study (2002) demonstrated an increased risk of renal cell carcinoma in males with occupational exposure to benzene, in a dose-response manner, with the excess risk being significantly associated to duration of exposure.[17] Compared with no exposure to the specific chemical, the adjusted OR was 1.8 (95% CI = 1.2–2.6).[17] Another occupational study of benzene exposure and kidney cancer was published by Greenland et al (1994). [18] This case-control study of benzene exposure in transformer manufacturing workers in Massachusetts found an OR of kidney cancer with benzene exposure of 4.29 (95% CI 1.33-13.8).[18] In addition, Seyyedsalehi et al (2024) performed a meta-analysis of 29 studies and found an association between occupational benzene exposure and kidney cancer, with an OR 1.20 (95% CI 1.03-1.39).[19]

I have read the general causation report of expert Dr. Benjamin Hatten and Dr. Steven Bird. These expert reports detail an extensive review of the epidemiology, toxicology and mechanism of action of PCE, VC and Benzene and kidney cancer. These reports are consistent with my review of the literature and support my opinions in this case.

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X. Epidemiology Specifically Related to Upper Tract Urothelial Carcinoma

I also reviewed epidemiological studies that specifically consider upper tract urothelial carcinoma either under the category of kidney cancer or separately from kidney cancer.[20,21,22,23,24] As stated above, many studies specifically list UTUC under the category of kidney cancer. For example, Zhao et al. conducted a cohort study of workers employed at an aerospace company between 1950 and 1993 who were exposed to TCE.[20] The authors constructed a job exposure matrix to assess exposure to TCE.[20] The authors classified cumulative exposure based on job title, and their time employed at each job title, as low, medium, high, or none.[20] The authors included malignant neoplasm of the renal pelvis—i.e. upper tract urothelial carcinoma—under the general category of kidney cancer.[20] A significant relationship between kidney cancer mortality and incidence was found at the high TCE exposure level with estimated risk ratios of 2.03 (95% CI: 0.50, 8.32) and 4.90 (95% CI: 1.23, 19.6), respectively.[20] At the medium TCE exposure level, there was an elevated risk of kidney cancer mortality and incidence with estimated risk ratios of 1.43 (95% CI: 0.49, 4.16) and 1.87 (95% CI: 0.56, 6.20), respectively.[20]

Similar results were reached by Pesch et al. when studying the relationship between chlorinated solvents and urothelial carcinoma.[21] In this study, upper tract urothelial carcinoma was considered under the category of urothelial carcinomas.[21] Long exposure to the job task of metal degreasing—which was considered an occupational setting with TCE exposure—had a significant relationship with incidence of urothelial carcinoma with an odds ratio of 2.3 (95% CI: 1.4-3.8).[21] An elevated risk of urothelial carcinoma was found even when the authors categorized exposure based on estimated exposure to select substances.[21] When assessing for select substance exposure using a job-task exposure matrix, the authors found an odds ratio of 1.8 (95% CI: 1.1-3.1) for substantial exposure to PCE and an odds ratio of 1.8 (95% CI: 1.2-2.7) for substantial exposure to TCE.[21]

Studies that categorized upper tract urothelial carcinoma separately reached similar results.[22,23] Raaschou-Nielsen et al. categorized renal pelvis cancer under the category of kidney cancer, but also separately analyzed the renal pelvis cancers alone. This study found an elevated risk of renal pelvis cancer for men exposed to TCE for at least three months with a SIR of 1.2 (95% CI: 0.81, 1.84).[22] The SIR for renal pelvis cancers was identical to the overall kidney cancer SIR, which was also 1.2 for men. Lynge et al. analyzed renal pelvis cancers separately from kidney cancer and found a significant relationship between both renal pelvis cancer and kidney cancer (analyzed separately) with benzene exposure for all Nordic countries: SIR of 2.0 (95% CI: 1.0-3.7) for renal pelvis cancer and SIR of 1.3 (95% CI: 1.0-1.7) for kidney cancer.[23]

In sum, renal pelvis cancer or UTUC has similar risk profiles when considered in the category of kidney cancer or when analyzed separately.[20,21,22,23,24] This supports the analyses used above and throughout this report.

XI. Impact of TCE, PCE, VC and Benzene Exposure from Camp Lejeune

The Agency for Toxic Substances and Disease Registry (ATSDR) has completed and reviewed several epidemiological studies and meta-analyses to determine if personnel and civilians were at increased risk for certain health effects from exposure to this contaminated water.[1] The evidence from the methodological studies establishes that exposure to the levels of the toxins in the drinking water at Camp Lejeune are causes of kidney cancer.[1] All meta-analyses that evaluated epidemiological studies of high utility were based on reports from agencies mandated to evaluate the health risk of the chemicals, including the IARC (2014), EPA (2011) or NTP (2015).[2,25,26] Interpretation of the findings in meta-analyses published and reviewed in the scientific literature for

TCE exposure and kidney cancer outline the magnitude of the adjusted Hazard Ratio (HR) between 1.2 and 1.4 across multiple studies, the precision of the effect estimates (CI>95%) and examine the impacts of unmeasured potential confounders and exposure misclassification on the HR estimate.[7,27] As noted, other studies in the literature have linked exposure to PCE, VC and benzene to the development of malignancies, including kidney cancer.

Based upon these studies and a literature review of occupational and environmental studies, the ATSDR report assessed the strength of the evidence supporting the causality of kidney cancer from TCE exposure.[1] The conclusion was that sufficient causal evidence exists linking TCE exposure and kidney cancer.[1] There was a monotonic exposure-response relationship between kidney cancer risk and TCE/ PCE exposure for Marines.[14]

There is additional epidemiologic literature relating specifically to the water at Camp Lejeune finding a causal relationship with kidney cancer/UTUC, including Bove 2014a, Bove 2014b, the ATSDR 2018 mortality study, the 2024 Bove mortality study and the 2024 Bove cancer incidence study.[8,9,10,14,28]

XII. The Levels of the Toxins in the Water at Camp Lejeune

ATSDR conducted historical reconstruction modeling to estimate the monthly average contaminant levels in the Tarawa Terrace (TT) and Hadnot Point (HP) distribution systems.[1] Median estimates from the HP distribution system during peak years for TCE was 366ug/L (range 0-783ug/L), PCE 15ug/L (range 0 to 39ug/L) and VC 22ug/L (range 0 to 67ug/L), all of which exceed the EPA's listing of the maximum contaminant level (MCL) for the volatile organic compounds in drinking water in the United States.[1] These values are 5 ug/L for TCE, PCE and benzene; 2 µg/L for vinyl chloride. In addition, the estimated drinking water concentrations of benzene consistently exceeded the current 5 ug/L MCL. This median estimate of TCE within the drinking water also exceeds median values observed to be associated with an increased risk of kidney cancer in several epidemiologic studies referenced within this report.[1]

There are three known exposure pathways from contaminated water: ingestion, inhalation and dermal absorption. Each pathway contributes to level of chemicals within the body, their known biological effects, and therefore to the overall cancer risk.

In reviewing the General Causation Expert Report of Benjamin Hatten, M.D, M.P.H, Dr. Hatten states "Given that the exposure of interest is water contaminated with multiple culprit compounds, the body of literature that directly examines the Camp Lejeune population exposed to the contaminated water system best answers the question of what levels of exposure are associated with kidney cancer." I agree with this statement, and it supports my opinions in this matter as to the causal connection between the camp Lejeune water and Mr. Mousser's kidney cancer.

Exposures to TCE, PCE, benzene and vinyl chloride at Camp Lejeune occurred simultaneously. TCE and PCE are Camp Lejeune water contaminants with a sufficient body of evidence for causation of kidney cancer, with non-monotonic exposure-relationships evident in studies involving Camp Lejeune.[8] Benzene and vinyl chloride are Camp Lejeune water contaminants with a body of evidence that meets the as likely as not standard for causation of kidney cancer. Therefore, an exposure to these compounds that is demonstrably hazardous to humans at Camp

Lejeune and is causally associated with kidney cancer is the lowest cumulative exposure category that demonstrates an elevated measure of association.

The RR for the cumulative exposure of each individual chemical as it was causally related to kidney cancer were as follows:

PCE: 1.40 (low exposures), 1.82 (medium exposures) and 1.59 (high exposures)[8]

VC: 1.66 (low exposures), 1.61 (medium exposures) and 1.51 (high exposures)[8]

Benzene: 1.31 (low exposure), 1.38 (medium exposures) and 1.36 (high exposures)[8]

TCE: 1.54 (low exposure), 1.21 (medium exposures) and 1.52 (high exposures)[8]

Dr. Hatten also states "the most relevant evidence for on-base exposures is a monotonic exposure-response relationship with TVOC rather than any individual component exposure (Bove 2014a). Thus, the lowest exposure category to cumulative TVOC with a monotonic dose-response provides evidence of a low level of Camp Lejeune water that is hazardous to human health and a known cause of kidney cancer." I agree with this statement and Dr. Hatten's report supports my opinions in this matter.

In Bove (2014a) the classification for low, medium and high exposures were:

TVOCs: >1 – 4600 ug/L-months (low exposure), >4600 – 12,250 ug/L-months (medium exposures) and >12,250 - 64,016 ug/L-months (high exposure)[8]

TCE: >1 – 3,100 ug/L-months (low exposure), >3,100 – 7,700 ug/L-months (medium exposure), >7,700 – 39,745 ug/L-months (high exposure)[8]

PCE: >1 – 155 ug/L-months (low exposure), >155 – 380 ug/L-months (medium exposure), >380 – 8,585 ug/L-months (high exposure)[8]

Vinyl chloride: >1 – 205 ug/L-months (low exposures), >205 – 500 ug/L-months (medium exposures), >500 – 2,800 ug/L-months (high exposures)[8]

Benzene: 2 – 45 ug/L-months (low exposures), >45 – 110 ug/L-months (medium exposures) >110 - 601 ug/L-months (high exposures)[8]

Mr. Mousser would have met the criteria for the high exposure category for each individual chemical at issue. For TVOC exposure, he was at the very upper end of the medium exposure, and just below the high exposure category.

The Camp Lejeune literature also analyzed exposure by time duration on base. A duration-based intensity of exposure is also supported by the Camp Lejeune literature with a monotonic exposure response evident.[10] The lowest duration category in the monotonic exposure-response finding that demonstrates an elevated measure of

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association is a level that is hazardous to human health and a known to cause kidney cancer. This is the "low" duration group with 1-5 quarters on base (HR 1.36).[10] Mr. Mousser would have been on base for multiples of this time period and would have been in the medium exposure category for this duration based assessment.

Dr. Hatten states in this report "To summarize, if an individual was present at Camp Lejeune and exposed to the levels of the chemicals above, this individual would have been exposed to levels of the water at Camp Lejeune that are hazardous to humans generally and are known to cause kidney cancer."

There were other levels shown in the literature that causally connect the toxins at issue in this case and kidney cancer. These were shown in the general causation reports for Drs. Hatten and Bird as well as cited elsewhere in this report. I will not repeat all these levels in this section, but all should be noted to be relevant to this analysis.

XIII. Specific Causation: TCE, PCE, VC and Benzene Exposure and Frank W. Mousser's Urothelial Cell Carcinoma of the Renal Pelvis

There are risk factors linked to the development of urothelial cell carcinoma of the renal pelvis. Those include tobacco use, exposure to chemicals and dyes used in manufacturing such as plastics, textiles and rubber, exposure to coal, tar, and asphalt, Balkan endemic nephropathy (BEN), genetic conditions such as Lynch syndrome, a history of transitional cell carcinoma of the bladder, use of cancer treating drugs cyclophosphamide and ifosfamide, and excessive use of phenacetin (a pain medication that hasn't been sold in the United States since 1983). An association between occupational risk factors and kidney cancer has also been established in several epidemiologic studies.[6] Occupations that have been linked to kidney cancer include the agricultural, dry cleaning and mechanical industries.

We employ scientific evidence, to attempt to ascertain whether exposure to the known carcinogens in the Camp Lejeune water was the cause of the Mr. Mousser's kidney cancer. Based upon the review of Mr. Mousser's medical records, his time stationed at Camp Lejeune, and review of the scientific and epidemiological evidence, it is my opinion that it is more likely than not that his exposure to the contaminated water at Camp Lejeune was the cause of his kidney cancer.

The following factors support my opinion:

(1) ATSDR historical reconstruction modeling to estimate the monthly average contaminant levels in the Tarawa Terrace (TT) and Hadnot Point (HP) distribution during the relevant times indicate that Mr. Mousser was exposed to water with TCE, PCE, Vinyl chloride and Benzene contamination levels exceeding carcinogenic levels observed in epidemiologic studies demonstrating an increased risk of kidney cancer associated with occupational or groundwater contamination TCE exposure, as well as PCE, VC and benzene exposure.[1]

(2) Frank W. Mousser was stationed at the French Creek Barracks for 891 days from October 18, 1982 through September 7, 1986, not including time away from the base for several deployments. During his time at camp Lejeune he lived at the French Creek Barracks supplied by the Hadnot Point water distribution system. The soldiers and civilian personnel at Camp Lejeune typically experienced multiple routes of exposure. In his deposition testimony, Mr. Mousser stated that he continued to eat and

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Bradford Hill factors provides evidence for causation that accounts for the principle of temporality, referring to the principle that the exposure of interest must have occurred prior to the development of the disease process of interest to be a cause.

2. [redacted]

3. [redacted]

4. [redacted]

5. [redacted]

XVII. Bradford Hill Factors

Multiple studies reviewed demonstrate an association between exposure to the contaminated Camp Lejeune water system and kidney cancer among Marines and civilians.[8,9,10,14,28] The Bradford Hill considerations are

employed for a structured analysis to determine whether this association with Mr. Mousser is causal, and specifically, whether that it is as likely as not that this exposure was the cause of Mr. Mousser's kidney cancer.

a. Strength of Association

Strength of association is demonstrated by statistical significance. Multiple studies discussed in this analysis demonstrate elevated measures of association between the Camp Lejeune water system that Frank Mousser was exposed to and kidney cancer.[8,9,10,28]

b. Consistency

Consistency refers to studies being done in different populations yielding similar results. Multiple cohort[8,9,10,28] and case control[14] studies reached similar conclusions, providing consistent evidence between an association between exposure to the water system at Camp Lejeune and kidney cancer.

c. Exposure-Response

Studies referenced in this report have demonstrated a monotonic exposure-response relationship between increased TVOC exposure and duration at Camp Lejeune.[8,28] This was a consistent finding despite varied methods of determining exposure within these studies. Frank Mousser, during his time at Camp Lejeune, was exposed to the levels of the chemicals listed above, and both his exposure levels to the individual toxins as well as total volatile organic compounds are hazardous to humans generally and are known to cause kidney cancer.

d. Temporality

Temporality refers to the principle that the exposure of interest must have occurred prior to the development of the disease process of interest to be a cause. Significant latency periods (10-20 years) were used in studies referenced in this report to ensure that the exposure to the Camp Lejeune water system occurred sufficiently prior the diagnosis of kidney cancer. [8,28] [redacted]

e. Biological Plausibility

This refers to the concept that a correlation between exposure and a disease process is causal based upon epidemiologic evidence. As discussed, TCE, PCE, vinyl chloride and benzene, all contaminants found in the water at Camp Lejeune, all meet the "as likely as not" standard for causation of kidney cancer. TCE and PCE have well documented mechanisms of kidney carcinogenesis, and vinyl chloride and benzene are both known carcinogens with biologically plausible mechanisms for causation of kidney cancer. The totality of the scientific evidence reviewed meets the biologic plausibility standard for Mr. Mousser's exposure to the Camp Lejeune water and kidney cancer.

f. Analogy

Frank Mousser's exposure to these toxins in the Camp Lejeune water system are analogous to other contaminated water systems that have been studied for association with kidney cancer, including two systems referenced in this report.[3,13] In addition, there is ample evidence of occupational exposures involving TCE, PCE, vinyl chloride and benzene that provide analogous evidence of causation to kidney cancer.

g. Specificity

The consideration of specificity is limited given that fact that the contaminants in the Camp Lejeune water system are known to cause other adverse health outcomes, including cancer in other organs. In addition, there are other unmodifiable and modifiable known risk factors to kidney cancer. As stated, [redacted] and his only known exposure was to the contaminants in the Camp Lejeune water system.

h. Coherence

The contaminants in the Camp Lejeune water system are known carcinogens, and literature reviewed includes mechanistic, human and animal studies that provide coherent data demonstrating the association between exposure to the water at Camp Lejeune and the development of kidney cancer.

i. Summary

When the abundant scientific and epidemiologic evidence that directly examines the Camp Lejeune water exposure and the development of Mr. Mousser's kidney cancer is considered through the Bradford Hill analysis, it is my conclusion that the exposure is more likely than not a cause of kidney cancer. Given Frank Mousser's known exposure to the Camp Lejeune water system, the levels found at Camp Lejeune during the relevant time period, and his lack of other risk factors, it is more likely than not to be the cause of his kidney cancer. This analysis helps put weight behind the causal relationship between the water at Camp Lejeune and Mr. Mousser's kidney cancer for purposes of the differential diagnosis and causal relationship.

XVIII. Mr. Mousser's Injuries

[redacted]